HYGIENE AND PUBLIC HEALTH

UNDER THE CHARGE OF

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A Note on Weil's Disease (Spirochetosis Icterohemorrhagica) as it Occurred in the Army in Flanders.—STOKES and RYLE report the following: Weil first described this disease in 1886. It was characterized by jaundice, pyrexia, hemorrhages and the fact that it was apparently infectious. Epidemics have been known in the United States, India, Africa, Japan and the near East. During the Gallipoli campaign there was a widespread epidemic of jaundice among the soldiers. As to the etiology of the disease, Inada and Ito, in 1914, reported the discovery of a spirochete in the liver of a guinea-pig which had been injected with the blood of a patient suffering from Weil's disease. These authors found later that the blood of patients recovering from Weil's disease contained protective substances against the spirochete they had found. They also showed that when they injected the blood of patients with Weil's disease during the first five days of the disease into the peritoneum of a guinea-pig the animal developed the symptoms of the disease and they were able to show the spirochete in the liver and blood of the animals in large numbers. The experiments made with normal people and patients with catarrhal jaundice showed no such results. These writers state that as the illness progresses the spirochetes disappear from the blood stream and cannot be demonstrated in the tissues. Weil believed that the infection was through the alimentary canal and Inada supports this view. Ito and Oki, however, have been able to communicate the disease to animals by applying infected material to the uninjured skin, and they therefore think it may be communicated to man by the skin being exposed to infective material. Fifteen cases of Weil's disease have come to the notice of the authors and they have been able to confirm the findings of the discoverers of the pathogenic cause of the disease. In 2 cases they have infected the animals which have shown the characteristic pathological changes and from which it has been possible to demonstrate the spirochetes. With the exception of 1 case all the men at the time of the onset of the disease were or recently had been employed in the trenches. Two of the cases proved fatal. The characteristic symptoms are as follows: The patients have generalized pains in the head and lower limbs, complain of weakness and unsteadiness. Most of them say they have been vomiting and some of them have nosebleed. The onset is accompanied by a temperature of 103° F. On admission the most striking symptoms were the jaundice and the extreme injection of the

conjunctivæ accompanied by great prostration. The authors found, in investigating the symptoms in the alimentary system, that the tongue is very dry, brown and fissured, that all the patients were constipated and vomiting in the early stages occurred in every case. The appetite which was lost during the period of pyrexia, returned as soon as the jaundice began to fade. As to the circulatory system, the pulse rate was, as a rule, slow in proportion to the pyrexia and there was a very definite slowing during convalescence. There was no evidence of respiratory complications. The excretory system was not greatly affected, the only change observed being a slight albuminuria during the pyrexial period in all cases. There were no severe nervous symptoms except in the grave cases. All the cases showed enlargement of the glands. Varying degrees of jaundice were seen. It was usually a lemon or orange tint and the color rapidly decreased as convalescence began. All cases showed an irregular pyrexia and subnormal temperature was common in early convalescence. In summarizing their experimental results the authors say that they had only two positive results among the animals injected with the blood of patients and no positive results were obtained after the sixth day. The infected guinea-pigs became ill between the fifth and seventh days. Their temperature rose to 104° F. and typical pathological changes identical with those described by the Japanese workers were demonstrated by a postmortem examination. Spirochetes were demonstrated in the livers in large numbers. In conclusion, the authors state that they believe the cause of epidemic jaundice in Flanders is identical with that described by the Japanese investigators and that the infective possibilities of the disease should be recognized.

The Etiology of Scarlet Fever.—MAIR (*Journal of Pathology and Bacteriology*) gives a preliminary description of the diplococcus scarlatinæ which he isolated from the throats of scarlet fever patients in 1915. In the present paper he gives a more complete description of this diplococcus with the technic employed for its isolation and identification. He also presents additional evidence in favor of its causal relationship to the disease. The organism can be demonstrated in the throats of scarlet fever patients during the first week of the disease in 87 per cent. of the cases and it disappears, as a rule, from the throat about the fifth week of convalescence. The diplococcus produces in monkeys a disease which in many respects resembles scarlet fever. It is confined to the region of primary invasion. The most outstanding clinical feature of scarlet fever, namely, the rash, has not been reproduced in monkeys.

Further Attempts to Transmit Pellagra to Monkeys.—EDWARD FRANCIS (*Hyg. Lab. Bull. No.* 106) states that the opinion of some workers that pellagra is a specific infectious disease, and the report by Harris, of New Orleans, of the production of pellagra in the monkey by the injection of a Berkefeld filtrate of pellagrous tissues, led the United States Public Health Service as a part of its study of pellagra to attack exhaustively the problem of the infectivity of pellagrous tissues and body fluids for the rhesus monkey. The experiments here reported consist of a series of inoculations and feedings of pella-

grous tissues and fluids into 90 rhesus monkeys, 3 baboons and 1 Java monkey. The experiments here recorded constitute the most exhaustive effort of its kind ever made to infect monkeys from pellagrous tissues. The experiments were begun at the United States Marine Hospital, Savannah, Ga., in July, 1913; were continued through 1914, and were completed in June, 1915. Throughout this period the experimental animals were bountifully fed and were kept under daily observation in cages in a glass-covered conservatory, located on the south side of the hospital and freely exposed to the direct rays of the sun. The sources of the pellagrous material with which the animals were inoculated or fed were 10 autopsies and 50 living pellagrins. In all, 252 experiments were made with material collected during life or at autopsy; in 141 of these, inoculations were made by hypodermic needle; in 82, material was fed by stomach-tube; in 29, feces were applied to the nasal mucosa. With few exceptions each animal was inoculated by more than one route, with two or more kinds of material, from more than one case, and on more than one occasion. Twenty-eight animals were each subjected to a single experiment; 19 were each subjected to two experiments; 17 were subjected to three experiments each; 19 were subjected to four each; 7 were subjected to five each; and 4 were subjected to six experiments each. The material used for inoculation or feeding was disposed of as follows: (*a*) The brain, spinal cord and their membranes were removed at 8 autopsies and injected cerebrally, venously, subcutaneously, muscularly, peritoneally or spinally into 29 rhesus monkeys. (*b*) The buccal, thoracic and abdominal organs except intestines were removed at one autopsy and injected venously or subcutaneously into 10 rhesus monkeys. (*c*) The intestines and fecal contents were removed at 7 autopsies and injected venously, cerebrally or subcutaneously into 18 rhesus monkeys, 2 baboons and 1 Java monkey. (*d*) Skin showing the pellagrous lesions was removed at 5 autopsies and injected cerebrally, venously or subcutaneously into 9 rhesus monkeys and 1 Java monkey. (*e*) Blood drawn from 8 pellagrins was injected venously, spinally, muscularly or peritoneally into 11 rhesus monkeys. (*f*) Cerebrospinal fluid collected at 5 autopsies was injected cerebrally, venously or spinally into 16 rhesus monkeys. (*g*) Spinal fluid collected during life from 28 pellagrins was injected immediately after collection, spinally, into 24 rhesus monkeys. (*h*) Pericardial fluid collected at 1 autopsy was injected venously or spinally into 4 rhesus monkeys. (*i*) Urine from 4 pellagrins giving a marked indican reaction was injected venously into 5 rhesus monkeys. (*j*) Feces from 3 pellagrins with marked diarrhea were after Berkefeld filtration injected peritoneally into 26 rhesus monkeys. (*k*) Feces from 1 autopsy and from 2 living pellagrins were introduced on cotton pledgets into the nasal fossæ of 29 rhesus monkeys. (*l*) Sputum collected fresh each day from 10 spitting pellagrins was fed by stomach-tube to 5 rhesus monkeys. (*m*) The brain, spinal cord and their membranes collected at 2 autopsies were fed by stomach-tube to 25 rhesus monkeys and 2 baboons. (*n*) The entire contents of the buccal and thoracic cavities collected at 2 autopsies were fed by stomach-tube to 16 rhesus monkeys and 3 baboons. (*o*) The entire contents of the abdominal cavity collected at 4 autopsies was fed by stomach-tube to 17 rhesus monkeys and 3

baboons. (*p*) The entire contents of the buccal, thoracic and abdominal cavities were removed at one autopsy, mixed with spoiled corn meal and fed by stomach-tube to 18 rhesus monkeys and 1 baboon. (*q*) Feces collected at 1 autopsy were mixed with spoiled corn meal and fed by stomach-tube to 2 rhesus monkeys. (*r*) Feces collected fresh each day from a fatal case of pellagra with diarrhea were mixed with spoiled corn meal and fed by stomach-tube daily for fifteen days to 6 rhesus monkeys. *Result:* The animals thus experimented upon showed no indications suggesting pellagra nor did they furnish any support for the view that pellagra is an infectious disease.

The Diagnosis of the Enteric Fevers in Inoculated Individuals by the Agglutinin Reaction.—DREYER and WALKER (*London Lancet*) believe that the agglutinin reaction is the most valuable method of diagnosis for the enteric fevers (typhoid, paratyphoid A and B), as the percentage of positive results obtained by culture methods alone does not exceed 50 per cent. In making the tests for agglutinins after inoculation some accurately quantitative method should be substituted for the qualitative methods formerly practised, diagnosis should depend upon a series of several successive observations and the method employed should produce definite and accurate measurements relative to some fixed standard: for instance, the standard agglutinable cultures prepared at Oxford. These methods would do away with the difficulties which arise from the wide practice of inoculation and would help to disprove the statement that protective inoculation against B. typhosus or the paratyphoid bacilli renders the agglutinin reaction less reliable than it is in non-inoculated individuals. The authors state that on the one hand active infection can be diagnosed as well in typhoid-inoculated individuals as in non-inoculated persons; and, on the other hand, the absence of inoculation agglutinins within the first twelve months or more after inoculation is a very rare occurrence in properly inoculated individuals and is no more frequent in the subjects of pyrexial attacks than in persons who remain in perfect health. The writers then attempt to show that the statements and methods of Dr. H. S. Tidy published in connection with a discussion of paratyphoid fever are erroneous. His method of comparison of the number of standard agglutinin units found in normal inoculated individuals with the number found in inoculated persons suffering from paratyphoid fever or other febrile condition is fallacious. His statement that inoculation agglutinins disappear or are diminished as the result of febrile attacks is shown to be disproved as well as his assertion that a serum titer of 100 standard agglutinin units "apparently corresponds to a positive reaction in non-inoculated persons." He claims also that from tables published by Dreyer and Torrens "it is evident that the febrile cases have distinctly less typhoid agglutinin than the normal inoculated persons," but the authors show that he has misinterpreted the data in these tables and has no true evidence to support this claim. Their contradiction of Tidy's statement that inoculation agglutinins disappear or are diminished as the result of febrile attacks is in perfect agreement with the careful work of Grattan, Harvey and Wood on paratyphoid fever in India. These workers showed that in undoubted cases of paratyphoid A fever in typhoid inoculated individuals a marked

increase in the agglutinins for B. typhosus was noted. The question as to whether inoculation-agglutinins are diminished after the fourth or fifth day of pyrexia can only be settled by following the serum titer of individuals during the course of an infection by exact quantitative measurements capable of reduction to n constant standard. From the study of a series of cases the writers found that although the typhoid agglutination titer may remain unchanged or only slightly increased during the course of paratyphoid fever, if affected at all, the change produced is one of active rise, either antecedent to or occurring at the same time as the beginning of the rise in parntyphoid B. agglutination titer. In diagnosing mild cases of typhoid and paratyphoid in inoculated persons, of which many occur, a quantitative determination of their agglutinin titer for typhoid and paratyphoid bacilli on three or more successive occasions at a few days' interval should be made before the presence of typhoid or paratyphoid infection can be excluded. In the interpretation of the agglutinin curves it is important to note the exact time of the maximum agglutinin titer, which should occur between the sixteenth and twenty-fourth day of the disease. If there is any deviation from this, careful inquiry should be made as to the accuracy of the dates, and the results should be carefully interpreted.

Anopheles Infectivity Experiments.—MITZMAIN (*Public Health Reports*, September 1, 1916, vol. xxxi, No. 35) states that in 17 experiments in which human beings were employed to test the infectibility of Anopheles punctipennis with Plasmodium vivax, 14 cases of malarial fever resulted. The sporozoites in the mosquitoes used developed ten to twenty-two days after the definite hosts were given an opportunity to bite a patient harboring a scanty number of mature tertian gametocytes. In an attempt to infect several persons with a single specimen of Anopheles punctipennis one mosquito proved to be the sole infective agent in 1 experiment and one proved to be the sole infective agent in 3 experiments. These two specimens when applied to the same person transmitted the infection in 5 cases, while one of them used with a third mosquito succeeded in infecting 4 persons. In these experimental inoculations it was demonstrated that in 9 instances in which two mosquitoes succeeded in transmitting malaria at least one of the pair was proved to be capable of causing the disease when used singly. It was demonstrated in 11 experiments that short exposure to bites was sufficient to cause successful transmission of the disease. In all of the successful inoculations only tertian infection was reproduced. Plasmodium vivax was demonstrated microscopically.

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